Subarachnoid Hemorrhage with Cerebral Vein Thrombosis and Aneurysmal Disease: A Treatment Conundrum

A Case Report

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Summary

Coexisting cerebral vein thrombosis and aneurysmal disease in the setting of acute subarachnoid hemorrhage is rare. We present the case of a 62 year-old woman presenting to our institution with an episode of collapse with a transient loss of consciousness with a 24 hour history of occipital headache. Imaging demonstrated extensive basal cisternal, intraventricular and cortical subarachnoid hemorrhage. Digital subtraction angiography and magnetic resonance imaging confirmed the presence of an aneurysm in the proximity of the right posterior communicating artery as well as thrombosis of the right anastomotic vein of Labbe. The patient underwent endovascular coil embolization to exclude the aneurysm and post-treatment anticoagulation. Despite a SAH rebleed with reversal of the anticoagulation, the patient subsequently made an unremarkable recovery with no neurological deficit.

Introduction

Coexisting cerebral vein thrombosis and aneurysmal disease in the setting of acute subarachnoid hemorrhage is exceedingly rare. Clinical management of the patient is such an instance is difficult. Determining the aetiology of the subarachnoid bleed is important as this consequently determines the treatment strategy; more specifically the initiation and degree of anticoagulation therapy balancing the risk of thromboembolism with further hemorrhage. The morbidity and mortality in such cases may be predictably high. We describe a case and discuss the basis of the management strategy in a patient who presents in this setting.

Case Report

A 62-year-old woman presented to our institution with an episode of collapse with a transient loss of consciousness with a 24 hour history of occipital headache. The patient was on hormone replacement therapy (HRT) and suffered from migraine but otherwise had no significant medical history. Glasgow Coma Scale (GCS) on presentation was 14/15 with no neurological deficit (WFNS grade 2). A coagulopathy screen was negative.

Computed Tomography (CT) (Figure 1) demonstrated extensive basal cisternal hemorrhage extending through the subarachnoid space of the right Sylvian fissure and cortical sulci with intraventricular blood (Fisher grade 4). Digital subtraction angiography (DSA) confirmed the presence of a right internal carotid artery (ICA) aneurysm close to the origin of the right posterior communicating artery (PComA). Segmental thrombosis of the right anastomotic vein of Labbe extending to its confluence with the transverse sinus was also ob-

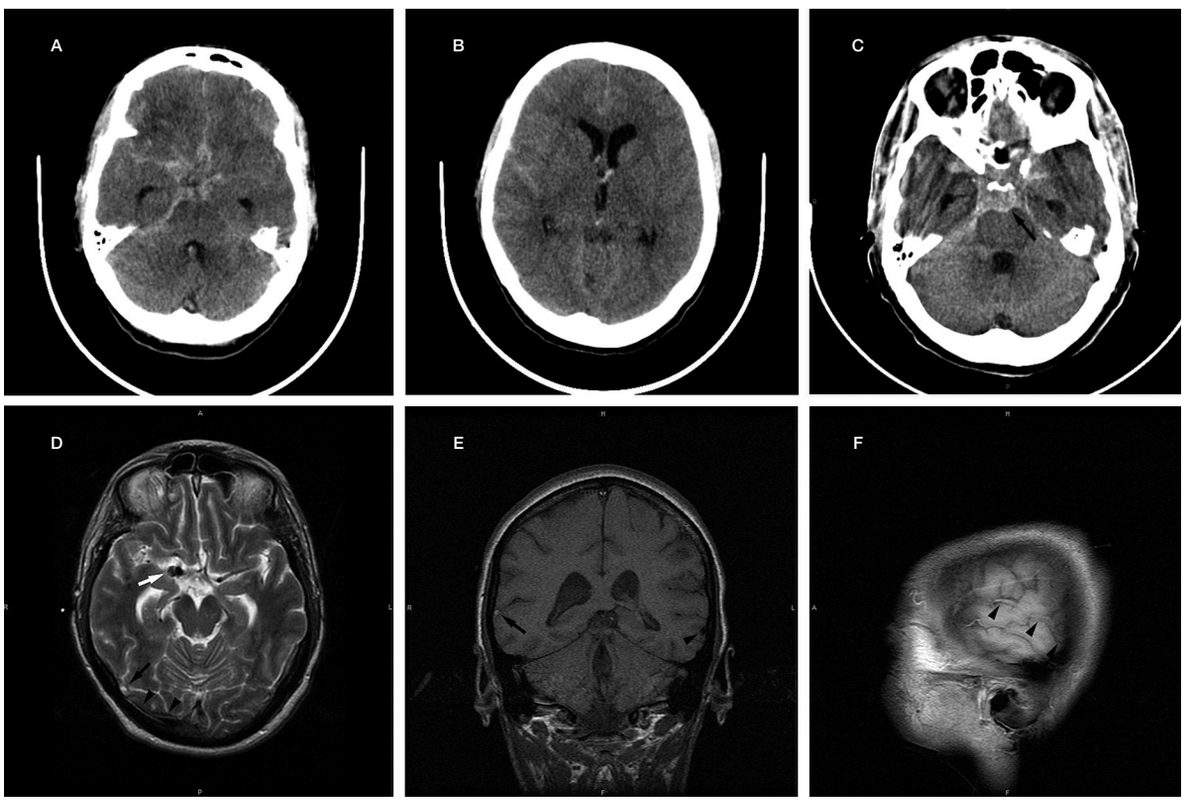


Figure 1 Uncontrasted axial CT images demonstrating (A) extensive acute subarachnoid blood particularly in the basal cisterns, (B) extending into ventricles and the cortical sulci of the right cerebral convexity. (C) A repeat uncontrasted CT study 12 hours post-coiling showing an acute rebleed, predominantly in the pontine cistern (arrow). Axial T2- (D), Coronal T1- (E) and Sagittal T1- (F) weighted MR acquisitions immediately post-coiling. D) Hyperintense signalled thrombus is seen at the confluence of the right anastomotic vein of Labbe (black arrow) with the right transverse sinus (arrowheads). Note the susceptibility artefact from the coiled right PComA aneurysm (white arrow). E) T1-weighted shortening of the right anastomotic vein of Labbe (arrow) in contradistinction to the signal within the left vein (arrowhead). F) T1-weighted shortening of the right anastomotic vein of Labbe on a right parasagittal section.

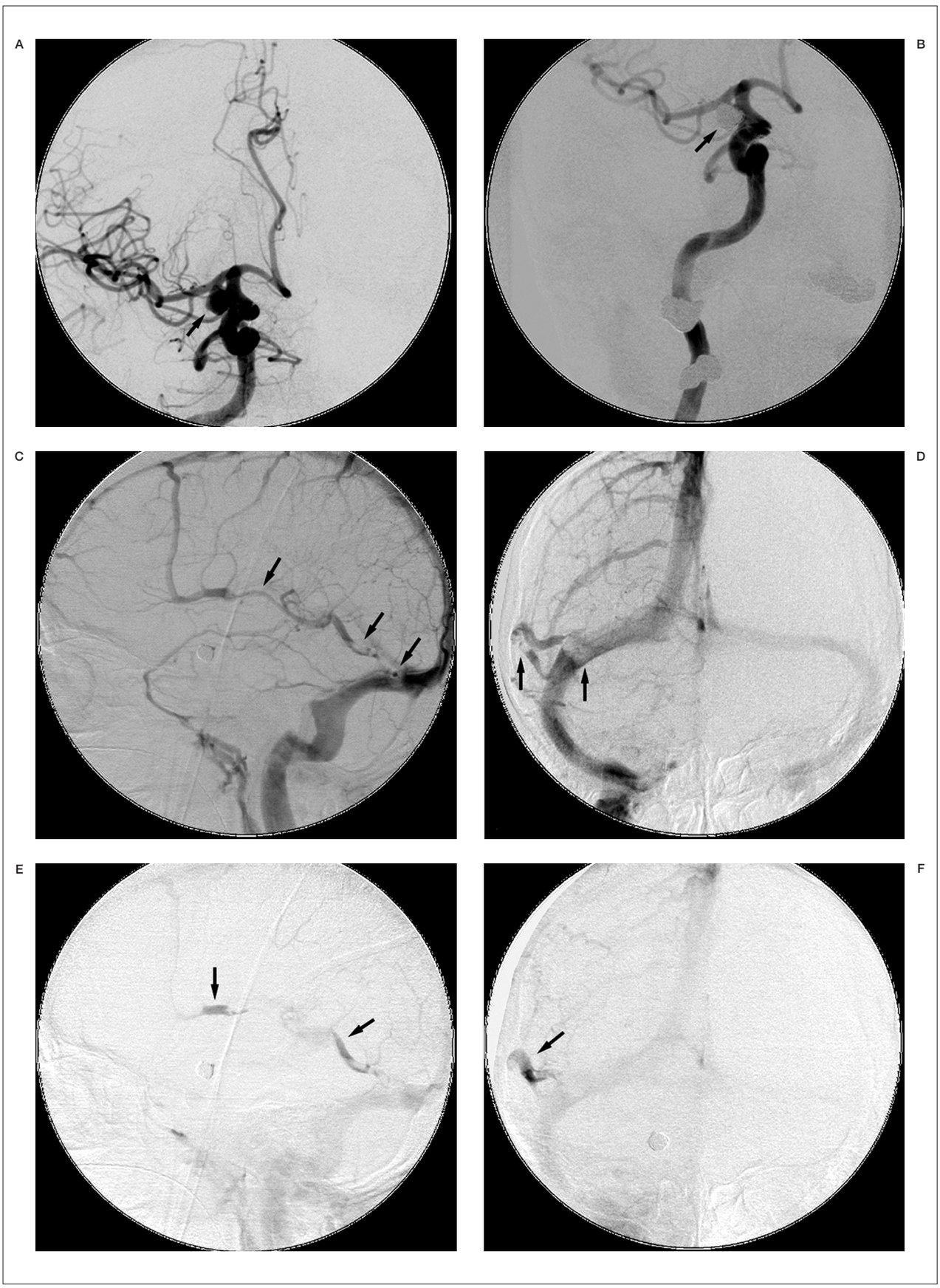
served with persistence of contrast within the vein in the delayed venous phase (Figure 2). This was subsequently confirmed on a magnetic resonance imaging (MRI) study (Figure 1). Endovascular coil embolization of the right ICA aneurysm was undertaken to full exclusion with planned post-procedural full heparin anticoagulation for 24 hours.

The patient experienced an episode of acute deterioration (GCS 3/15) during the recovery period which was confirmed to be acute hemorrhage particularly within the pontine cistern, despite good exclusion of the aneurysm (Figure 1). The heparinisation was reversed, and an external ventricular drain (EVD) inserted. The patient subsequently made an uneventful recovery and was discharged two weeks later with no neurological sequelae.

Discussion

Both intracranial aneurysms and venous thrombosis (cortical and sinus) are recognised causes of subarachnoid hemorrhage (SAH) [1,2,3]. Spontaneous SAH is related to a ruptured aneurysm in 85% of cases. However, SAH can even present as the initial manifestation of cere-

Figure 2 Procedural DSA images demonstrating: A) the right PComA aneurysm pre-embolization in the early arterial phase, and B) the excluded coiled right PComA aneurysm in the early arterial phase. Lateral and frontal projections respectively of (C and D) segmental filling defects representing thrombus (arrows) within the right anastomotic vein of Labbe, extending to its confluence with the right transverse sinus in the late venous phase. E,F) Delayed venous phase in which persistent contrast stasis was seen oscillating within the vein between the thrombosed segments.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bral venous thrombosis (CVT). In most of such cases the distribution of SAH is over the cortical surface of the brain, typically associated with a cortical venous infarction which may or may not be hemorrhagic. It is clear that venous hemorrhagic infarction is responsible for the production of SAH by secondary rupture into the subarachnoid space, however a mechanism of CVT with secondary venous hypertension resulting in the rupture of fragile, thin-walled cortical veins has been proposed [4].

It is therefore important to look for the presence of venous thrombosis on a CTA, particularly if the SAH is over the cortical surface. When both entities coexist in an acute SAH, the established cause will determine firstly the necessity and urgency in treating the aneurysm and secondly, treatment of the venous thrombosis by anticoagulation to prevent clot propagation or rarely clot thrombolysis [5].

Despite both the aneurysm and CVT occurring on the same side as the bleed, the pattern of distribution of blood made acute aneurysm rupture most likely in our patient. HRT was a possible risk factor for developing CVT in our normally ambulatory patient. Interestingly, raised intracranial pressure resulting from venous thrombosis has been postulated to cause venous dilatation with subsequent rupture [3] or aneurysmal rupture [1]; the latter remains a possible aetiological factor in our patient.

Endovascular coil embolization of the aneurysm was thus undertaken, with subsequent heparin anticoagulation to attempt to prevent thrombus propagation. However, optimal treatment of CVT remains controversial partly due to the variable nature of disease progression, further complicating the management of such patients [6]. Heparinisation was reversed after our patient acutely deteriorated after developing a further subarachnoid bleed, but subsequently made an uneventful recovery.

In a report of a similar case by Carvi y Nievas et Al, despite successful clipping of a ruptured anterior communicating artery aneurysm, the patient propagated thrombus into the superior sagittal and transverse sinuses and subsequently died despite moderate anticoagulation post-surgery [1].

Conclusions

Our case clearly illustrates both the difficulty in establishing the cause of subarachnoid hemorrhage in the presence of both aneurysmal and CVT disease as well as the initiation of post-endovascular coil embolization anticoagulation therapy. Based on our case and selected cases in the literature, it would appear that instigating anticoagulation treatment with subsequent close clinical, imaging and transcranial pressure monitoring is to be advocated.

References

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